skin tyrosinase [28] and are 10 times less than those reported for hamster [4,5,25] and mouse [7,15] melanoma tyrosinase, suggesting that there is a difference between normal melanocyte tyrosinase and abnormal neoplastic melanocyte tyrosinase. Apparent K_m for dopa as cofactor was $0.71–1.08 \times 10^{-6}$ M for TPA tyrosinase and $0.62–1.44 \times 10^{-6}$ M for normal tyrosinase (Table III). These values are the same as that of hamster melanoma tyrosinase [9]. The determination of the K_m for dopa with hamster melanoma tyrosinase required a correction for the lag in tyrosine oxidation at low dopa concentration [9], but this was not required in the present study. With the assay conditions used with hairbulb tyrosinase, there appeared to be no significant lag with the dopa concentrations used, and all plots of activity over time passed through or very close to zero for all dopa concentrations.

In summary, these studies show that some of the properties of TPA hairbulb tyrosinase are the same as normal hairbulb tyrosinase and that this enzyme does not appear to have altered kinetics. The defect with this form of albinism must represent a mutation involving some other step in melanin synthesis such as substrate transport or inhibitor production. Studies are underway to evaluate these possibilities.

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Announcement

The American Dermatological Society for Allergy and Immunology will meet September 28–30, 1978, at the Airport Hilton, Denver, Colorado.